

Original Research Article

Comparison of Rosuvastatin (10mg) and Atorvastatin (10mg) in achieving the treatment goals of dyslipidemia: Government Medical College, Suryapet**Polagani Padma¹, C. Muralidhar², Kavitha Mudavath^{3*}**¹Associate Professor, Department of Pharmacology, Government Medical College, Suryapet, Telangana, India.²Assistant Professor, Department of Pharmacology, Government Medical College, Suryapet, Telangana, India.³Associate Professor, Department of Pharmacology, Government Medical College, Nalgonda, Telangana, India.

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Abstract

Background: Atherosclerosis and coronary artery disease are both caused by dyslipidemia, which has been identified as the primary cause. The statin medication class is the most often recommended therapy for dyslipidemia. Among these, atorvastatin and rosuvastatin, a relatively new medication, are more commonly recommended. The goal of the study was to evaluate the efficacy of rosuvastatin and atorvastatin in the treatment of dyslipidemia. **Methods and Material:** Patients with dyslipidemia between the ages of 30 and 72 were eligible. One of two therapy groups was allocated to the patients. A total of 250 patients were randomly divided into two groups, each with 125 patients, and labeled Group I and Group II. For 12 weeks, Group I was given rosuvastatin (10 mg tablet OD) while Group II was given atorvastatin (10 mg tablet OD). The lipid profile low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), non-high-density lipoprotein cholesterol (non-HDL-C), triglycerides (TG), and very-low-density lipoprotein cholesterol (VLDL-C) were measured before the start of therapy and after 12 weeks. Percentage changes from baseline were calculated and adverse effects were recorded. **Results:** During the 11 months study, A total of 250 patients were screened for the study, of which males represented 63.60% (n = 159), female 36.40% (91) of the population, male and female ratio was 1.7:1. A total of 250 patients were divided randomly into two groups of 125 patients each and assigned as group I (male 80, female 30) and Group II (male 79, female 31). The group-I mean age was 53.66 ± 7.69 and group-II mean age 52.75 ± 6.89 . Group I received rosuvastatin (10 mg tablet OD) and Group II received atorvastatin (10 mg tablet OD) for 12 weeks. The levels of serum TC and LDL-C are decreased by 38.01% and 47.55 % respectively with the use of rosuvastatin (Group-I) after 12 weeks. atorvastatin 10 mg/day ((Group-II) for 12 weeks resulted in a statistically significant fall in levels of serum TC and LDL-C by 23.50 % and 29.79 %. **Conclusions:** Generally, medication of Rosuvastatin could reach a better lipid-reducing effect and yield a higher attainment rate of LDL-C and TC than Atorvastatin in the same dose in high-risk hyperlipidemic Indian patients.

Keywords: Rosuvastatin, Atorvastatin, Dyslipidemia, Treatment goals.

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Introduction

A large body of data suggests that reducing LDL C (low-density lipoprotein cholesterol) lowers the risk of cardiovascular disease (CVD) [1, 2]. The use of 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) as first-line treatment for dyslipidemia is recommended in both European and US CVD prevention recommendations, and target LDL-C values are specified [3,4]. For particularly high-risk individuals, a National Cholesterol Education Program (NCEP) study recently suggested lowering target values to even more aggressive LDL-C targets [5, 6]. Despite the demonstrated advantages of LDL-C lowering, many patients fail to meet suggested LDL-C targets [7, 8]. The use of medicines with poor LDL-C lowering effectiveness and inadequate dosage titration is the most likely causes. Goal attainment is particularly low in high-risk patients with increased LDL-C because therapy with larger dosages of statins is frequently required to achieve their target LDL-C levels [9, 10]. Furthermore, these patients are given more aggressive LDL-C targets, which are more difficult to meet. The most effective statin at the lowest dose would be a straightforward, successful treatment plan that would allow more people to meet their goals without having

to adjust their dosage. At a dosage of 10 mg, rosuvastatin has shown great effectiveness in reducing LDL-C, allowing patients with hypercholesterolemia to meet their lipid objectives [11-13]. Furthermore, rosuvastatin has favorable effects on other lipid profile components, such as high-density lipoprotein cholesterol (HDL-C), which is a substantial, independent risk factor for CVD [14, 15].

Out of these drugs, rosuvastatin and atorvastatin are commonly prescribed drugs for dyslipidemia. Rosuvastatin is a relatively newer member of statins which is more expensive than existing members of statins and its prescription is also on the rise. That is why in the present study rosuvastatin has been included. Atorvastatin has been taken as a standard drug because it is more frequently and has been reported to be a better drug in dyslipidemia over other members of the statin group. All of these factors have formed the basis of the present study in which the effect of rosuvastatin and atorvastatin on lipid profile has been compared in addition to their safety in dyslipidemic patients.

Materials and Methods

A prospective observational study was carried out in the department of orthopedics of Government Medical College, Suryapet, for eleven months (January 2019 to November 2019), after obtaining the institutional ethical permission

Selection criteria: A total of 250 patients between the ages of 30-72 years old with an aberrant lipid profile (serum total cholesterol >200 mg%, LDL-C >100 mg %, or HDL-C 35 mg %) were included in the study at Government Medical College, Suryapet, Medical

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OPD/Ward. After thoroughly describing the research protocols to the patients, they gave their permission.

Study Design

A hospital-based study.

Study Setting

Sample Size:

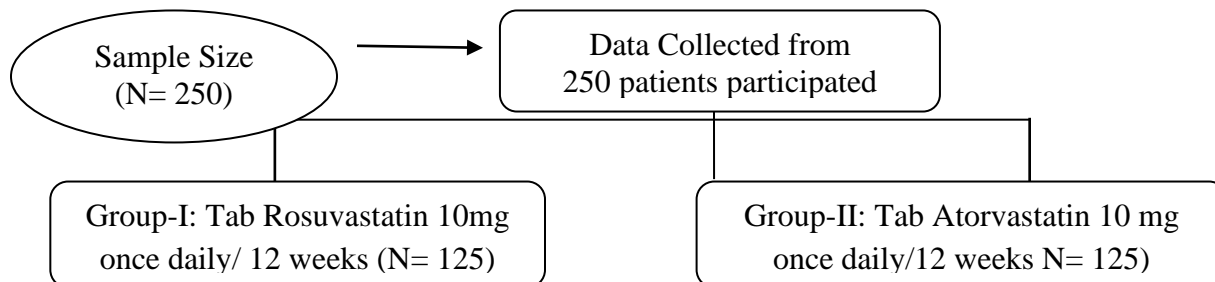
Government Medical College, Suryapet.

Study Period

January 2019 to November 2019.

Data Collection

By using a pre-designed, pretested questionnaire.



Exclusion Criteria

Patients are excluded from the study if they are:

1. Hypersensitive to rosuvastatin or atorvastatin.
2. Having diabetes mellitus/ hypothyroidism/ nephrotic syndrome/ Gout/ pancreatitis / uncontrolled hypertension.
3. Pregnant and lactating women.
4. Women on oral contraceptive pills (OCPs) estrogen/hormone replacement therapy.
5. Having a history of muscle pain (fibromyalgia with associated raised CPK levels).
6. Patients with abnormal liver function test (LFT) and renal function test (RFT).
7. Concurrently using CYP3A4 isoenzyme i.e.azole antifungals, macrolides, calcium channel blockers (except dihydropyridines e.g. nifedipine, amlodipine), cyclosporine, histamine-2 blockers, grapefruit juice, and enzyme inducers like phenobarbitone, rifampicin, phenytoin, carbamazepine.

Design of the Study

The research was conducted in a randomized, open-label, and parallel fashion. After obtaining the first baseline, overnight fasting cholesterol levels at the start of the study (week 0), and subsequent values in the 12th week of the research, the results were analyzed. Following enrolment, individuals' medical histories were obtained and a physical examination was performed. A total of 250 patients were divided randomly into two groups of 125 patients each and assigned as a group I and Group II. Group I received rosuvastatin (10 mg tablet OD) and Group II received atorvastatin (10 mg tablet OD) for 12 weeks. In both groups, patients were instructed to take drugs 30 minutes before an evening meal. The patients were advised to continue with their dietary modification and physical activity and they have explained the schedule of the drug treatment. The patients were also advised to report immediately in case they developed unexplained muscle pain/undue tiredness, low urine output, or any other symptoms about side effects of the drugs. At the end of the study, the patients were kept under regular follow-up to monitor adverse events for additional 6 weeks with the Department of Pharmacology Government Medical College, Suryapet.

Parameters and Laboratory Procedures

The study was carried out by first documenting each patient's baseline examinations and then repeating them at the 12th week. Patients were also told to return to the hospital after 6 weeks to check any adverse reactions and to report any adverse events at any time.

At each patient visit, the patient's history, clinical examination, and adverse events were documented, and blood samples were taken for lipid profile estimate at the 0 week and 12th week following a 12-hour overnight fast. The research was done at the Department of Pharmacology with the use of proprietary kit techniques. A spectrophotometer was used to analyze the blood samples. After obtaining samples, serum was tested using a centrifuge machine that rotated at 2000 revolutions per minute for 10 minutes at 37°C, and serum was used to evaluate TC levels, followed by HDL-C levels after VLDL-C and LDL-C precipitation. The CHOD/POD-phosphotungstate technique was used to test blood samples using a spectrophotometer [16, 17]. Very low-density lipoprotein cholesterol (VLDL-C) was calculated by formula [18]

$$\text{VLDL-C} = \text{Triglycerides}/5$$

Low-density lipoprotein cholesterol (LDL-C) was calculated by using Friedewald's formula [18]

$$\text{LDL-C (mg/dl)} = \text{T.C (mg/dl)} - \text{HDL-C (mg/dl)} - (\text{Triglycerides}/5)$$

Non-HDL-C was calculated by: TC – HDL-C.

Statistical Analysis

Data generated from the study was evaluated and expressed as the mean \pm SD of each variable. Paired Student's test was applied within the group after the treatment interval and an unpaired test was applied when 2 groups were compared. All statistical analysis was performed using Origin Pro 8.5 statistical software.

Results

During the 11 months study, A total of 250 patients were screened for the study, of which males represented 63.60% (n = 159), female 36.40% (91) of the population, male and female ratio was 1.7:1. A total of 250 patients were divided randomly into two groups of 125 patients each and assigned as group I (male 80, female 30) and Group II (male 79, female 31). The group-I mean age was 53.66 ± 7.69 and group-II mean age 52.75 ± 6.89 . Group I received rosuvastatin (10 mg tablet OD) and Group II received atorvastatin (10 mg tablet OD) for 12 weeks. Table -1 showing Lipid levels (mg/dl) goals proposed for Asian-Indians.

Group I and II Lipid levels (mg/dl) in Indian patients before and after 12 weeks of therapy (Fig-1 and 2).

Lipid levels (mg/dl) and comparative percentage changes in Indian patients before and after 12 weeks of therapy results were showing table-2.

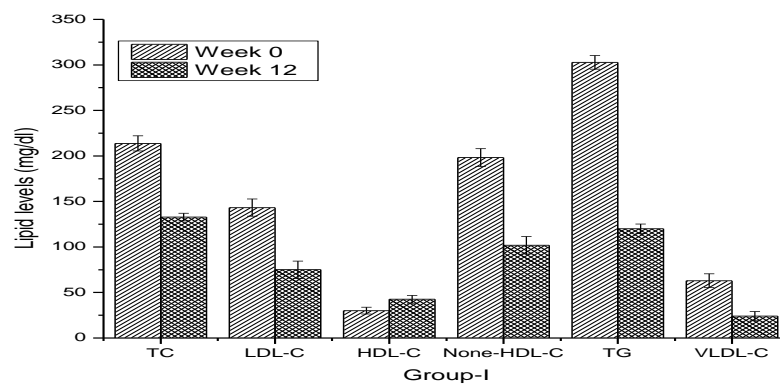
Table 1: Lipid levels (mg/dl) goals proposed for Asian-Indians [19]

	LDL-C	TC	Non-HDL-C
Optimum	<80	<150	<110
Near or above optimal	80-99	150-169	110-129
Borderline high	100-114	170-184	130-144
High	115-129	185-199	145-159
Very high	>130	>200	>160

Table 2: Lipid levels (mg/dl) and comparative percentage changes in Indian patients before and after 12 weeks of therapy.

Lipids/lipoproteins		Group I Mean \pm SD (N=125)	Group II Mean \pm SD (N=125)	Change between Group I and II
TC	Week 0	213.80 \pm 8.26	213.7 \pm 6.26	
	Week 12	132.82 \pm 4.17	163.23 \pm 7.72	
	% change	38.01	23.50	14.51*
	p value	<0.001	<0.001	<0.001
LDL-C	Week 0	143.23 \pm 9.42	141.16 \pm 7.37	
	Week 12	75.03 \pm 9.49	99.1 \pm 8.53	
	% change	47.55	29.79	17.76*
	p value	<0.001	<0.001	<0.001
HDL-C	Week 0	30.1 \pm 3.71	31.16 \pm 4.12	
	Week 12	42.6 \pm 4.30	41.5 \pm 4.21	
	% change	40.00	32.25	7.75*
	p value	<0.001	<0.001	<0.001
Non-HDL-C	Week 0	198.26 \pm 9.79	197.66 \pm 5.43	
	Week 12	101.85 \pm 9.50	109.86 \pm 6.73	
	% change	49.00	44.67	4.33*
	p value	<0.001	<0.001	<0.001
TG	Week 0	302.83 \pm 7.43	303 \pm 9.68	
	Week 12	120 \pm 5.16	136.5 \pm 5.54	
	% change	60.26	55.12	5.14*
	p value	<0.001	<0.001	<0.001
VLDL-C	Week 0	62.96 \pm 7.48	63.4 \pm 7.93	
	Week 12	24 \pm 5.03	30.6 \pm 6.10	
	% change	61.30	52.39	8.91*
	p-value	<0.001	<0.001	<0.001

*Difference in percentage change between group I and group II; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; TG, triglycerides; Lp, lipoprotein; Apo, apolipoprotein. *p-value obtained from analysis of variance comparing rosuvastatin 10 mg with atorvastatin for percentage change in lipid and lipoproteins.

**Fig 1: Lipid levels (mg/dl) of Group-I in Indian patients before and after 12 weeks of therapy**

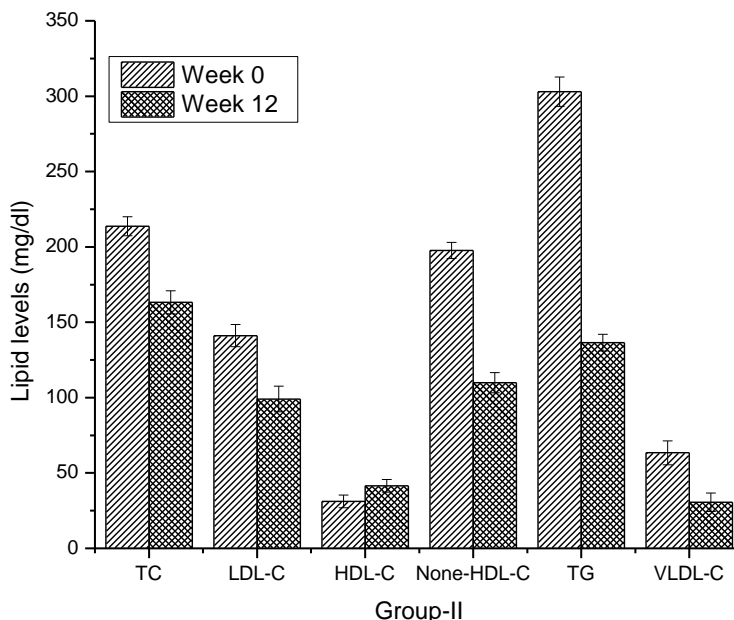


Fig 2: Lipid levels (mg/dl) of Group-II in Indian patients before and after 12 weeks of therapy

Discussion

In a patient with CAD, dyslipidemia is defined as TC > 200 mg/dl, LDL-C > 100 mg/dl, TG > 150 mg/dl, and HDL-C < 40 mg/dl [20]. The existence of one or more risk factors determines the values of these indicators in non-CAD patients. Dyslipidemia is one of the primary risk factors for the development of CAD, and the goal of dyslipidemia medication therapy is to bring these parameters' values into the desired range. Currently, statins are the most often recommended medications to treat this condition. In several comparative clinical trials, atorvastatin was shown to be superior to simvastatin, which was reported to be superior to the other members of the family [21, 22].

Both HMG-CoA reductase inhibitors, atorvastatin, and rosuvastatin were examined as monotherapy in individuals with dyslipidemia in this research. Both of these medications have been found to have a significant impact on blood TC and LDL-C levels.

Group I and Group II with 125 patients each participated in the study with the average age for group I being, 53.66 ± 7.69 years and for group II 52.75 ± 6.89 years.

At the start of the research, the mean blood total cholesterol levels were 241.62 29.57 mg/dl. These values were substantially higher than Gandhi's reported mean TC levels of 157.29 mg/dl in a sample of 201 healthy metropolitan Delhi individuals [23]. These levels were also greater than those reported by Gopinath *et al.*, [24], who found 210 mg/dl and 169 mg/dl in CAD patients in urban and rural regions, respectively. The mean blood HDL-C values reported at the start of the research were 31.63 5.9 mg/dl. These values were lower than those published by Gopinath *et al.*, [23], who found serum HDL-C levels of 56.13 mg/dl in urban subjects and 51.09 mg/dl in rural subjects, as well as levels reported by Gupta *et al.*, [25], who found greater levels in rural males than in urban men (44.0 13 vs 43.1 12 mg/dl). At the start of the research, the mean blood LDL-C and TG values were 145.19 24.89 and 325.91 88.55 mg/dl, respectively. VLDL-C levels at the start of the research were 65.18 17.7 mg/dl on average.

Group I: After 12 weeks, serum TC and LDL-C levels are reduced by 38.01 percent and 47.55 percent, respectively, when rosuvastatin is used (Table-2). This decrease in TC is greater than that reported by Blasetto *et al.* [26], who reported a 34 percent decrease in TC. However, when analyzing the effect of rosuvastatin on levels of TG and HDL-C, an extremely significant fall of 60.91 percent in TG levels and a rise of 29.26 percent in HDL-C levels was reported after 12 weeks, almost identical to 48.1 percent as reported by Blasetto *et al.*, [26] and 48 percent as reported by Ballantyne *et al.*, [27]. This change in levels was significantly higher than 28.8% and 12.9% respectively as reported by Blasetto *et al.*, [26] who also stated that patients with elevated TG appeared to have a greater percentage decrease in TG levels and greater percentage increase in HDL-C than do those with lower TG. This change in levels of TG and HDL-C was also significantly higher than 23% and 10% respectively as reported by Ballantyne *et al.*, [27]

Group II: In the present study atorvastatin 10 mg/day for 12 weeks resulted in a statistically significant fall in levels of serum TC and LDL-C by 23.50 % and 29.79 % (Table -2). This fall was lower than 33% and 42% as reported by Nosedá *et al.*, [28] with 10mg/day of atorvastatin at 12 weeks. This fall is almost similar to the fall reported by Mckenney *et al.*, [29] who reported a fall of 26% and 30% for serum TC and LDL-C with the use of atorvastatin 10 mg/day for 12 weeks. Atorvastatin decreased the serum TG by 50.75% in 12 weeks which significantly more than 25% decrease in serum TG after 12 weeks of use of atorvastatin as reported by Tiek C. Ooi *et al.*, [30] On analyzing the effect of atorvastatin on serum VLDL-C, it was found that serum VLDL-C decreased by 50.75% at 12 weeks. This fall at 12 weeks was higher than the fall as reported by

Tiek C. Ooi *et al.*, [30] reported a fall of 35% in VLDL-C levels after 12 weeks of administration of atorvastatin 10 mg/day. The rise in levels of serum HDL-C by atorvastatin at the end of 12 weeks was 32.15%. This rise is significantly more than 10% as reported by Frost *et al.*, [31]

Both drugs were well tolerated. Nausea/vomiting, headache, and myalgia (mild muscle pain) were reported in few patients in both groups but the difference was not significant. Apart from these, no other side effect was noticed in 12 weeks of the study.

Rosuvastatin is a significantly more potent blocker of hepatocyte sterol than all other statins were currently available. It differs structurally from other statins, containing a polar methane sulphonamide group which confers relative hydrophilicity, which in turn imparts greater selectivity for uptake into hepatic versus non-hepatic cells [32].

CONCLUSION:

Generally, medication of Rosuvastatin could reach a better lipid-reducing effect and yield a higher attainment rate of LDL-C and TC than Atorvastatin in the same dose in high-risk hyperlipidemic Indian patients. The study afforded additional evidence supporting superior therapeutic efficacy of Rosuvastatin over Atorvastatin.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee of Government Medical College, Suryapet Telangana, India.

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Conflict of Interest: Nil

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